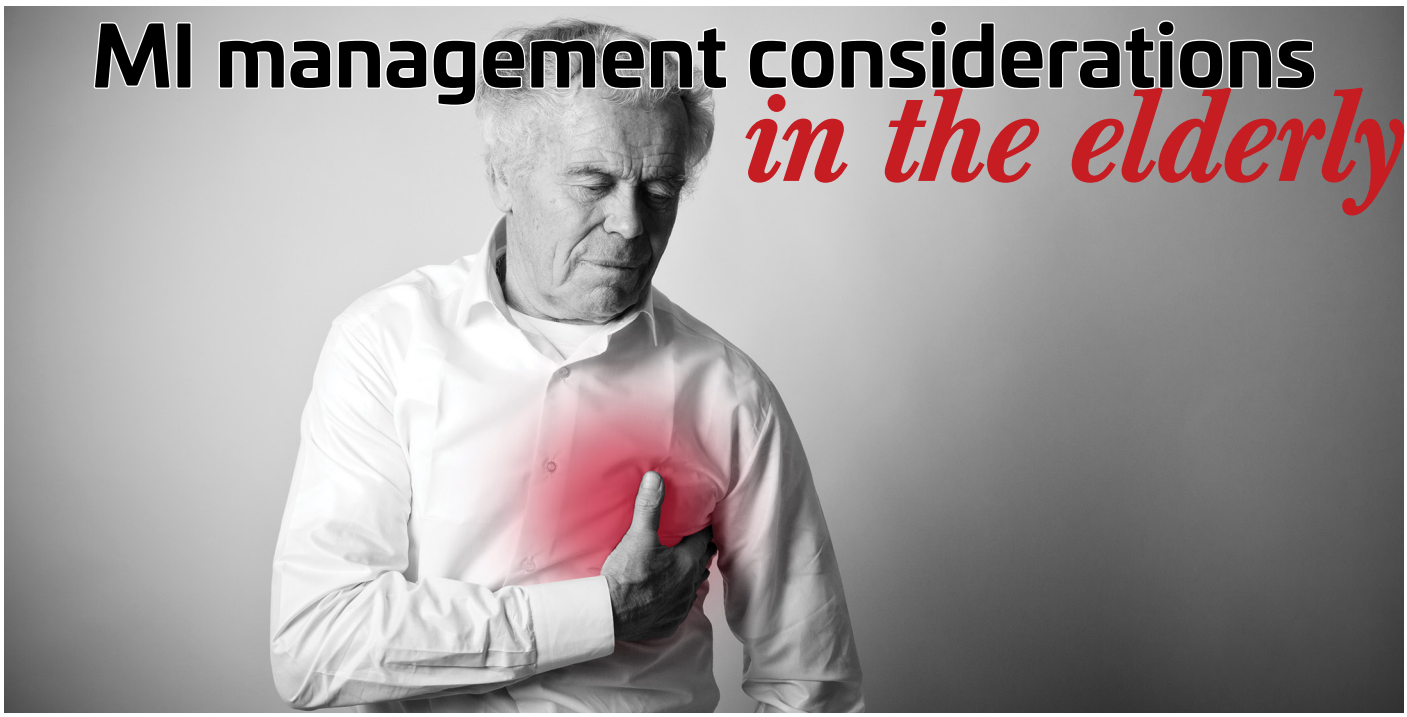
MI management considerations in the elderly



One of the acknowledged problems is that the elderly has not been represented enough in randomised clinical trials of myocardial infarction (MI) management. Now, 40 years since the first percutaneous coronary intervention (PCI) was performed, we still do not know the optimal revascularisation strategy in elderly patients. On the positive side, it has also been shown that the mortality of acute myocardial infarction (AMI) has declined due to evidence-based therapies and life-style changes.

General issues in myocardial infarction

An acute MI surely is the most severe form of coronary artery disease with a high risk of mortality and recurrence and the risk can be reduced with evidence-based therapy. More than a third of deaths in developed countries are due to MIs.

AMIs are divided into ST-elevated MI (STEMI) and non-ST elevated MIs (NSTEMI-STEMI). NSTEMI currently is more common than STEMI. Together they are considered acute coronary syndrome (ACS), while unstable angina is also an ACS with similar pathophysiology than NSTEMI and therefore they are together referred to as non-ST-segment elevated ACS (NSTE-ACS). Since the use of high-sensitivity troponin measurements many unstable anginas have been reclassified as NSTEMI.

A classic MI, in most cases, is due to disruption of a vulnerable (unstable) atherosclerotic plaque or an erosion of the plaque. It is much more common in these cases to find a coronary artery obstruction of 30%-50% as compared to coronary artery obstruction of ≥70%.

A vulnerable plaque has a thin fibrous cap, lots of lipids in the core together with many inflammatory cells. After disruption of such a plaque there develops a platelet clot followed by a blood clot, which suddenly completely or partially occludes the coronary artery. A STEMI by implication has a completely obstructed coronary artery. This hypercoagulable state could then contribute to the rupture of other vulnerable plaques so that can be more than one culprit lesion causing the MI.

Management principles

Critical in the management of a MI is the concept to reperfuse the obstructed coronary artery, stabilise the vulnerable plaque and to stop the hypercoagulable state and prevent further clotting by platelets and clotting factors. All who survive must be put on secondary prevention therapy to reduce the risk of death and recurrence.

Reperfusion in STEMI

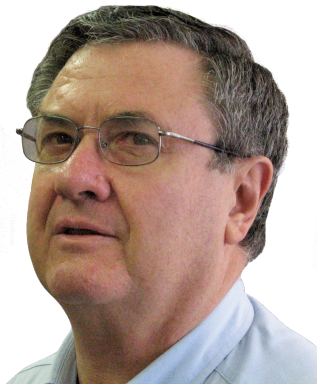
1 It is essential to restore coronary artery patency, using PCI as primary therapy, within 60-90 minutes, preferably less than 60 minutes from onset of symptoms. Stenting with a drug-eluting stent is necessary in most to reduce the residual artery stenosis, which will further improve perfusion and prevent acute reocclusion. Rapid reperfusion is associated with smaller infarct size and lower mortality as compared to those patients who experienced a delay in reperfusion. Where a primary PCI cannot be done, transfer to a capable hospital is necessary and PCI can be done with time less than 120 minutes from symptom onset. During PCI thrombus aspiration is not routinely done but kept for specific indications as deemed necessary by the cardiologist during the PCI.

2 Thrombotic agents promote the conversion of plasminogen to plasmin which then lyse fibrin and dissolves the blood clot. Fibrinolysis does reduce mortality but is inferior to primary PCI. Fibrinolysis can be important where PCI is not available and then must only be given for STEMI as it causes more harm than benefit in NSTEMI. In most cases patients need to be transferred for PCI after fibrinolysis.

Reperfusion in NSTEMI

- The decision in these patients is to decide an initial invasive strategy with PCI or coronary artery

Elderly people represent a group in the community that is rapidly increasing in absolute numbers. Elderly patients, especially those older than 80 years have a high prevalence of coronary artery disease, which is not unexpected.



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bypass grafting within about 48 hours or early conservative medical therapy with PCI for those with recurrent myocardial ischaemia and by the patient's risk.

Medical therapy in myocardial infarction

- Aspirin reduces death and recurrent MI as compared to placebo. Loading dose is 162mg-325mg as soon as possible followed by indefinite maintenance of 75mg-100mg daily. The lower dose is as effective with less bleeding than the high maintenance dose of aspirin.
- The P2Y12 inhibitors, which antagonise the ADP-receptor on the platelet, such as clopidogrel is added to aspirin for all who underwent a PCI with stenting in a dose of 300mg loading and followed by maintenance dose of 75mg daily. Randomised clinical trials have shown added mortality benefit in ACS. Dual-antiplatelet therapy (DAPT) should be continued for at least one year after an ACS regardless whether they received PCI with stent or medically managed. DAPT therapy reduces stent thrombosis but could also reduce recurrent events. Some advice on the preferred use of more powerful P2Y12 antagonists such as ticagrelor and prasugrel (prasugrel not for elderly people older than 75 years). Benefit does come with the price of increased bleeding. Triple therapy with two antiplatelet drugs and oral anti-coagulant can improve outcome with increased risk of bleeding.
- Anti-coagulants: Low-molecular weight heparin such as enoxaparin, reduce mortality in ACS especially given to NSTEMI or unstable angina.
- Statins are given routinely in ACS. Indications for beta-blockers, renin-angiotensin system blocker and aldosterone inhibitors improve outcome in selected patients. These are all considered necessary in secondary prevention as well.

Management in the elderly

With these general recommendations for managing ACS and MI, one immediately asks the question on how these principles are applied to the elderly and very elderly? Age remains a significant confounding factor that leads to reduced adherence to guideline-directed therapies and invasive strategies for ACS.

In a retrospective real-world observational study in the United States using data from the largest in-patient database from 1998 to 2013, data were available on 6 720 281 hospitalisations with ACS in patients ≥70 years of age. The aim was to report temporal trends and outcomes of this elderly group of patients.

Only 18.3% of these elderly patients with ACS received a PCI. There was an upward trend in the percentage of elderly with any type of ACS who received a PCI: 9.4% in 1998 vs 28.3% in 2013. This increasing trend for PCI in the elderly was also seen in STEMI and in NSTEMI patients and this increasing trend was seen in all age categories (70-79, 80-89 and >90 years).

Despite an increase in comorbidities in the elderly presenting with an ACS, the mortality rate in those elderly who received a PCI declined over time. In this 16 year very large group of elderly ACS patients, there was an increase in PCI use with a concomitant reduction in mortality. PCI is independently associated with lower mortality in elderly patients with ACS.

In a randomised clinical trial, elderly patients with coronary artery disease (ACS, angina, silent ischaemia), who had a stenosis of at least 70% of a coronary artery, was randomised to a drug-eluting stent or bare-metal stent. (SENIOR trial). The primary endpoint was a composite off: Major adverse cardiac and cerebrovascular events (mortality, MI, stroke or revascularisation).

It was shown in elderly patients that the use of PCI with drug-eluting stents and a shorter time on DAPT was significantly better than Bare-metal stent and a similar duration of DAPT. The shorter DAPT duration is attractive in the elderly due to less bleeding complications.

There is an increasing issue of frailty in the elderly with ACS. Frailty is associated with an increased mortality, especially in women. In the elderly with frailty there is some evidence that is associated with event rates and possibly stent thrombosis.

Conclusions

There is a paucity on outcome data in the elderly when presenting with an ACS and treated with or without a PCI as initial therapy.

Very large real-world data has shown an increase in the use of PCI for the treatment of ACS in the elderly with a result that mortality rates have decreased despite an increase in the number of elderly patients who also have more co-morbidity.

Frailty seems to confer a higher risk of an adverse event when the elderly present with an ACS and then treated with a PCI. Data however is scanty.

It seems that there is a lack of the use of guideline-directed therapy when the elderly present with ACS. The elderly benefit when guideline-directed therapy is applied including the use of PCI as directed by guidelines.

You are never too old to get the best guideline-directed treatment for ACS.

References available on request. SF